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L1 and trail	3

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Set Name Query

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result set

DB=PGPB,USPT; PLUR=YES; OP=OR

<u>L3</u>	L1 and trail	3	<u>L3</u>
<u>L2</u>	L1 and (trail and antiprogestin)	0	<u>L2</u>
<u>L1</u>	Kumar-vijay.in.	52	<u>L1</u>

END OF SEARCH HISTORY

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? s androgen(5n)(independen? or insensitiv?)
    100035 ANDROGEN
    1549213 INDEPENDEN?
    117085 INSENSITIV?
S1 11511 ANDROGEN(5N)(INDEPENDEN? OR INSENSITIV?)
? s trail(5n)resistan?
    19960 TRAIL
    1740950 RESISTAN?
S2 1448 TRAIL(5N)RESISTAN?
? s s1 and s2
    11511 S1
    1448 S2
S3 7 S1 AND S2
? rd

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>>>Duplicate detection is not supported for File 340.

>>>Records from unsupported files will be retained in the RD set.

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S4 3 RD (unique items)
? t s4/3,k,ab/1-3

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4/3,K,AB/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)
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15595825 PMID: 15897871

Methylseleninic acid sensitizes prostate cancer cells to TRAIL-mediated apoptosis.

Yamaguchi Kenya; Uzzo Robert G; Pimkina Julia; Makhov Peter; Golovine Konstantin; Crispen Paul; Kolenko Vladimir M

Department of Urological Oncology, Fox Chase Cancer Center, Philadelphia, PA 19111, USA.

Oncogene (England) Sep 1 2005, 24 (38) p5868-77, ISSN 0950-9232--
Print Journal Code: 8711562

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

Tumor necrosis factor-related apoptosis-inducing ligand (TRAIL) is a cytotoxic agent that preferentially induces apoptosis in a variety of human cancer cells. Unfortunately, some tumor cells remain ***resistant*** to TRAIL-mediated cell death. Therefore, agents that sensitize malignant cells to TRAIL-mediated cell death might be of particular importance for the development of novel antitumor therapeutic regimens. Recent studies establish a critical role of selenium in prostate cancer prevention in vitro and in vivo. Here, we demonstrate that concomitant administration of TRAIL and methylseleninic acid (MSA) produces synergistic effects on the induction of apoptosis in androgen-dependent LNCaP and androgen - ***independent*** DU-145 prostate cancer cells. MSA rapidly and specifically downregulates expression of the cellular FLICE inhibitory protein, a negative regulator of death receptor signaling. In addition, we demonstrate that the synergistic effects of MSA and TRAIL result from the activation of the mitochondrial pathway-mediated amplification loop. Addition of MSA effectively blocked TRAIL-mediated BAD phosphorylation at Ser112 and Ser136 in DU-145 cells and was accompanied by induction of the mitochondrial permeability transition and release of apoptogenic cytochrome c and Smac/DIABLO proteins from the mitochondria and into the cytosol. These results suggest that selenium-based dietary compounds may help to overcome resistance to TRAIL-mediated apoptosis in prostate cancer cells.

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4/3,K,AB/2 (Item 2 from file: 155)
DIALOG(R) File 155:MEDLINE(R)
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13490190 PMID: 11759069

Actinomycin D and gemcitabine synergistically sensitize androgen-***independent*** prostate cancer cells to Apo2L/TRAIL-mediated apoptosis. Zisman A; Ng C P; Pantuck A J; Bonavida B; Belldegrun A S
Department of Urology, UCLA School of Medicine, University of California at Los-Angeles, 90095, USA.

Journal of immunotherapy (Hagerstown, Md. - 1997) (United States)
Nov-Dec 2001, 24 (6) p459-71, ISSN 1524-9557--Print Journal Code: 9706083

Publishing Model Print

Document type: Journal Article

Languages: ENGLISH

Main Citation Owner: NLM

Record type: MEDLINE; Completed

The cytotoxic efficacy and kinetics involved in sensitization of Apo2L/TRAIL-resistant, androgen-independent prostate cancer cells to Apo2L/TRAIL or tumor necrosis factor-alpha or Fas ligand-mediated apoptosis were tested using subclinical concentrations of actinomycin D, paclitaxel, cisplatin, gemcitabine, and radiation in CL-1, LNCaP, DU-145, and PC3 prostate cancer cell lines. CL-1 cells expressed all four Apo2L/TRAIL receptors and were resistant to Apo2L/TRAIL-mediated apoptosis (1-5,000 ng/mL) and to the sensitizers when given alone. Pretreatment with actinomycin D followed by Apo2L/TRAIL or tumor necrosis factor-alpha or anti-Fas CH-11 monoclonal antibody, but not in the reverse order, induced apoptosis in all cell lines. Synergistic sensitization in CL-1 cells was shown also with gemcitabine but not with cisplatin, VP-16, paclitaxel, or radiation. Incubating the Apo2L/TRAIL-resistant CL-1, LNCaP, DU-145, and PC3 cell lines with 100 ng/mL actinomycin D for 4 hours followed by Apo2L/TRAIL for 24 hours resulted in 45.4 +/- 10.3%, 58.8 +/- 3.6%, 53.4 +/- 1.4%, and 84.2 +/- 8.4% apoptosis, respectively. Prolonging the sensitization time to 24 hours followed by 20 hours of incubation with Apo2L/TRAIL further enhanced the killing activity against CL-1 cells to 89 +/- 1% (delta = 60%, synergistic ratio = 3.1). This killing has a biphasic pattern that was contributed to by apoptosis (83%) and necrosis (17%) at 10 hours (peak) and 40% and 60%, respectively, at 20 hours. These results suggest that prostate cancer cells' resistance to Apo2L/TRAIL-mediated apoptosis can be reversed and synergy is achieved by sensitization of tumor cells with subclinical concentrations of actinomycin D or gemcitabine and may be useful clinically for the treatment of metastatic hormone- and drug-refractory prostate cancer.

Actinomycin D and gemcitabine synergistically sensitize androgen-***independent*** prostate cancer cells to Apo2L/TRAIL-mediated apoptosis. The cytotoxic efficacy and kinetics involved in sensitization of Apo2L/TRAIL-resistant, androgen-independent prostate

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4/3,K,AB/3 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci
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12895715 Genuine Article#: 831NY Number of References: 41

Title: X-linked inhibitor of apoptosis protein inhibition induces apoptosis and enhances chemotherapy sensitivity in human prostate cancer cells (ABSTRACT AVAILABLE)

Author(s): Amantana A; London CA; Iversen PL; Devi GR (REPRINT)

Corporate Source: AVI Biopharma Inc,Canc & Endocrine Program,4575 SW Res Way,Suite 200/Corvallis//OR/97333 (REPRINT); AVI Biopharma Inc,Canc & Endocrine Program,Corvallis//OR/97333(grdevi@avibio.com)

Journal: MOLECULAR CANCER THERAPEUTICS, 2004, V3, N6 (JUN), P699-707

ISSN: 1535-7163 Publication date: 20040600

Publisher: AMER ASSOC CANCER RESEARCH, 615 CHESTNUT ST, 17TH FLOOR, PHILADELPHIA, PA 19106-4404 USA

Language: English Document Type: ARTICLE

Abstract: apoptosis and enhanced chemotherapy sensitization in

androgen -refractory prostate cancer cells. ***Androgen*** - insensitive prostate cancer cells are highly resistant to several chemotherapeutic drugs and are characterized by the appearance of apoptosis-resistant cells. In this study, we identified the critical role of X-linked inhibitor of apoptosis protein (XIAP), a potent antiapoptotic factor, in conferring chemotherapy resistance in an ***androgen*** - ***insensitive*** DU145 human prostate cancer cell line. Results reveal that DU145 cells were highly resistant to cisplatin, but this resistance was overridden when the cells were treated for a prolonged time (>96 hours) with cisplatin (IC50 = 27.5 to 35.5 mumol/L). A decrease in levels of XIAP and Akt/phospho-Akt and an increase in caspase-3 activity were identified to be key factors in cisplatin sensitivity (40% to 55% decrease in cell viability) at later time points. In contrast, tumor necrosis factor-related apoptosis-inducing ligand (TRAIL) treatment caused a 40% to 50% decrease in cell viability within 6 hours (IC50 = 135 to 145 ng/mL). However, increasing concentrations or prolonged treatment with TRAIL did not change drug potency. A significant increase in caspase-3 activity was observed with TRAIL treatment with no apparent change in XIAP levels. Specific inhibition of XIAP expression using an antisense XIAP phosphorodiamidate morpholino oligomer induced apoptosis and increased caspase-3 activity. Combination of cisplatin with XIAP antisense potentiated cisplatin sensitivity by decreasing the IC50 from > 200 mumol/L with cisplatin alone to 9 to 20 mumol/L and decreasing incubation time required for activity from 96 to 24 hours. Similarly, TRAIL in combination with XIAP antisense phosphorodiamidate morpholino oligomer enhanced TRAIL potency by 12- to 13-fold. In conclusion, abrogation of XIAP expression is essential for therapeutic apoptosis

and enhanced chemotherapy sensitization in androgen-refractory prostate cancer cells.

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that DU145 cells were highly resistant to...

...Identifiers--LIGAND-MEDIATED APOPTOSIS; C-MYC; UP-REGULATION; XIAP;
RESISTANCE; EXPRESSION; TRAIL; LNCAP; GENE; IAP

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